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Office of Pollution Prevention and Toxics
Environmental Protection Agency
401 M Street., S.W.
Washington, D.C. 20460
Attn: Section 8(e) Coordinator (CAP Agreement)

Dear Coordinator:

8ECAP-0025

On behalf of the Regulatee and pursuant to Unit II B.1.b. and Unit II C of the 6/28/91CAP Agreement, E.I. Du Pont de Nemours and Co. hereby submits (*in triplicate*) the attached studies. Submission of this information is voluntary and is occasioned by unilateral changes in EPA's standard as to what EPA now considers as reportable information. Regulatee's submission of information is made solely in response to the new EPA §8(e) reporting standards and is not an admission: (1) of TSCA violation or liability; (2) that Regulatee's activities with the study compounds reasonably support a conclusion of substantial health or environmental risk or (3) that the studies themselves reasonably support a conclusion of substantial health or environmental risk.

The "Reporting Guide" creates new TSCA 8(e) reporting criteria which were not previously announced by EPA in its 1978 Statement of Interpretation and Enforcement Policy, 43 Fed Reg 11110 (March 16, 1978). The "Reporting Guide states criteria which expands upon and conflicts with the 1978 Statement of Interpretation. Absent amendment of the Statement of Interpretation, the informal issuance of the "Reporting Guide" raises significant due processes issues and clouds the appropriate reporting standard by which regulated persons can assure TSCA Section 8(e) compliance.

For Regulatee,

Mark H. Christman
Counsel
Legal D-7158
1007 Market Street
Wilmington, DE 19898
(302) 774-6443

ATTACHMENT 1

Submission of information is made under the 6/28/91 CAP Agreement, Unit II. This submission is made voluntarily and is occasioned by recent changes in EPA's TSCA §8(e) reporting standard; such changes made, for the first time in 1991 and 1992 without prior notice and in violation of Regulatee's constitutional due process rights. Regulatee's submission of information under this changed standard is not a waiver of its due process rights; an admission of TSCA violation or liability, or an admission that Regulatee's activities with the study compounds reasonably support a conclusion of substantial risk to health or to the environment. Regulatee has historically relied in good faith upon the 1978 Statement of Interpretation and Enforcement Policy criteria for determining whether study information is reportable under TSCA §8(e), 43 Fed Reg 11110 (March 16, 1978). EPA has not, to date, amended this Statement of Interpretation.

After CAP registration, EPA provided the Regulatee the June 1, 1991 "TSCA Section 8(e) Reporting Guide". This "Guide" has been further amended by EPA, EPA letter, April 10, 1992. EPA has not indicated that the "Reporting Guide" or the April 1992 amendment supersedes the 1978 Statement of Interpretation. The "Reporting Guide" and April 1992 amendment substantively lowers the Statement of Interpretation 's TSCA §8(e) reporting standard[2]. This is particularly troublesome as the "Reporting Guide" states criteria, applied retroactively, which expands upon and conflicts with the Statement of Interpretation.[3] Absent amendment of the Statement of Interpretation, the informal issuance of the "Reporting Guide" and the April 1992 amendment clouds the appropriate standard by which regulated persons must assess information for purposes of TSCA §8(e).

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[2]In sharp contrast to the Agency's 1977 and 1978 actions to soliciting public comment on the proposed and final §8(e) Policy, EPA has unilaterally pronounced §8(e) substantive reporting criteria in the 1991 Section 8(e) Guide without public notice and comment, See 42 Fed Reg 45362 (9/9/77), "Notification of Substantial Risk under Section 8(e): Proposed Guidance".

[3]A comparison of the 1978 Statement of Interpretation and the 1992 "Reporting Guide" is a appended.

Throughout the CAP, EPA has mischaracterized the 1991 guidance as reflecting "longstanding" EPA policy concerning the standards by which toxicity information should be reviewed for purposes of §8(e) compliance. Regulatee recognizes that experience with the 1978 Statement of Interpretation may cause a review of its criteri. Regulatee supports and has no objection to the Agency's amending reporting criteria *provided that* such amendment is not applied to the regulated community in an unfair way. However, with the unilateral announcement of the CAP under the auspices of an OCM enforcement proceeding, EPA has wrought a terrific unfairness since much of the criteria EPA has espoused in the June 1991 Reporting Guide and in the Agency's April 2, 1992 amendment is new criteria which does not.exist in the 1978 Statement of Interpretation and Enforcement Policy.

The following examples of new criteria contained in the "Reporting Guide" that is not contained in the Statement of Interpretation follow:

o even though EPA expressly disclaims each "status report" as being preliminary evaluations that should not be regarded as final EPA policy or intent[4], the "Reporting Guide" gives the "status reports" great weight as "sound and adequate basis" from which to determine mandatory reporting obligations. ("Guide" at page 20).

o the "Reporting Guide" contains a matrix that establishes new numerical reporting "cutoff" concentrations for acute lethality information ("Guide" at p. 31). Neither this matrix nor the cutoff values therein are contained in the Statement of Interpretation. The regulated community was not made aware of these cutoff values prior to issuance of the "Reporting Guide" in June, 1991.

othe "Reporting Guide" states new specific definitional criteria with which the Agency, for the first time, defines as 'distinguishable neurotoxicological effects'; such criteria/guidance not expressed in the 1978 Statement of Interpretation.[5];

othe "Reporting Guide" provides new review/ reporting criteria for irritation and sensitization studies; such criteria not previously found in the 1978 Statement of Interpretation/Enforcement Policy.

othe "Reporting Guide" publicizes certain EPA Q/A criteria issued to the Monsanto Co. in 1989 which are not in the Statement of Interpretation; have never been published in the Federal Register or distributed by the EPA to the Regulatee. Such Q/A establishes new reporting criteria not previously found in the 1978 Statement of Interpretation/Enforcement Policy .

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[4]The 'status reports' address the significance, if any, of particular information reported to the Agency, rather than stating EPA's interpretation of §8(e) reporting criteria. In the infrequent instances in which the status reports contain discussion of reportability, the analysis is invariably quite limited, without substantial supporting scientific or legal rationale.

[5] *See, e.g,* 10/2/91 letter from Du Pont to EPA regarding the definition of 'serious and prolonged effects' as this term may relate to transient anesthetic effects observed at lethal levels; 10/1/91 letter from the American Petroleum Institute to EPA regarding clarification of the Reporting Guide criteria.

In discharging its responsibilities, an administrative agency must give the regulated community fair and adequate warning to as what constitutes noncompliance for which penalties may be assessed.

> Among the myriad applications of the due process clause is the fundamental principle that statutes and regulations which purport to govern conduct must give an adequate warning of what they command or forbid.... Even a regulation which governs purely economic or commercial activities, if its violation can engender penalties, must be so framed as to provide a constitutionally adequate warning to those whose activities are governed.

Diebold, Inc. v. Marshall, 585 F.2d 1327, 1335-36 (D.C. Cir. 1978). *See also*, Rollins Environemntal Services (NJ) Inc. v. U.S. Environmental Protection Agency, 937 F. 2d 649 (D.C. Cir. 1991).

While neither the are rules, This principle has been applied to hold that agency 'clarification', such as the Statement of Interpretation, the "Reporting Guide" nor the April 1992 amendments will not applied retroactively.

> ...a federal court will not retroactively apply an unforeseeable interpretation of an administrative regulation to the detriment of a regulated party on the theory that the post hoc interpretation asserted by the Agency is generally consistent with the policies underlying the Agency's regulatory program, when the semantic meaning of the regulations, as previously drafted and construed by the appropriate agency, does not support the interpretation which that agency urges upon the court.

Standard Oil Co. v. Federal Energy Administration, 453 F. Supp. 203, 240 (N.D. Ohio 1978), aff'd sub nom. Standard Oil Co. v. Department of Energy, 596 F.2d 1029 (Em. App. 1978):

The 1978 Statement of Interpretation does not provide adequate notice of, and indeed conflicts with, the Agency's current position at §8(e) requires reporting of all 'positive' toxicological findings without regard to an assessment of their relevance to human health. In accordance with the statute, EPA's 1978 Statement of Interpretation requires the regulated community to use scientific judgment to evaluate the significance of toxicological findings and to determining whether they reasonably support a conclusion of a substantial risk. Part V of the Statement of Interpretation urges persons to consider "the fact or probability" of an effect's occurrence. Similarly, the 1978 Statement of Interpretation stresses that an animal study is reportable only when "it contains reliable evidence ascribing the effect to the chemical." 43 Fed Reg. at 11112. Moreover, EPA's Statement of Interpretation defines the substantiality of risk as a function of both the seriousness of the effect and the probability of its occurrence. 43 Fed Reg 11110 (1978). Earlier Agency interpretation also emphasized the "substantial" nature of a §8(e) determination. See 42 Fed Reg 45362, 45363

(1977). [Section 8(e) findings require "extraordinary exposure to a chemical substance...which critically imperil human health or the environment"].

The recently issued "Reporting Guide" and April 1992 Amendment guidance requires reporting beyond and inconsistent with that required by the Statement of Interpretation. Given the statute and the Statement of Interpretation's explicit focus on substantial human or environmental risk, whether a substance poses a "substantial risk" of injury requires the application of scientific judgment to the available data on a case-by-case basis.

If an overall weight-of-evidence analysis indicates that this classification is unwarranted, reporting should be unnecessary under §8(e) because the available data will not "reasonably support the conclusion" that the chemical presents a substantial risk of serious adverse consequences to human health.

Neither the legislative history of §8(e) nor the plain meaning of the statute support EPA's recent lowering of the reporting threshold that TSCA §8(e) was intended to be a sweeping information gathering mechanism. In introducing the new version of the toxic substances legislation, Representative Eckhart included for the record discussion of the specific changes from the version of H. R. 10318 reported by the Consumer Protection and Finance Subcommittee in December 1975. One of these changes was to modify the standard for reporting under §8(e). The standard in the House version was changed from "causes or contributes to an unreasonable risk" to "causes or significantly contributes to a substantial risk". This particular change was one of several made in TSCA §8 to avoid placing an undue burden on the regulated community. The final changes to focus the scope of Section 8(e) were made in the version reported by the Conference Committee.

The word "substantial" means "considerable in importance, value, degree, amount or extent". Therefore, as generally understood, a "substantial risk" is one which will affect a considerable number of people or portion of the environment, will cause serious injury and is based on reasonably sound scientific analysis or data. Support for the interpretation can be found in a similar provision in the Consumer Product Safety Act. Section 15 of the CPSA defines a "substantial product hazard" to be:

> "a product defect which because of the pattern of defect, the number of defective products distributed in commerce, the severity of the risk, or otherwise, creates a substantial risk of injury to the public."

Similarly, EPA has interpreted the word 'substantial' as a quantitative measurement. Thus, a 'substantial risk' is a risk that can be quantified, *See*, 56 Fed Reg 32292, 32297 (7/15/91). Finally, since information pertinent to the exposure of humans or the environment to chemical substances or mixtures may be obtained by EPA through Sections 8(a) and 8(d) regardless of the degree of potential risk, §8(e) has specialized function. Consequently, information subject to §8(e) reporting should be of a type which would lead a reasonable man to conclude that some type action was required immediately to prevent injury to health or the environment.

Attachment

Comparison:

Reporting triggers found in the 1978 "Statement of Interpretation/ Enforcement Policy",43 Fed Reg 11110 (3/16/78) and the June 1991 *Section 8(e) Guide.*

TEST TYPE	1978 POLICY CRITERIA EXIST?	New 1991 GUIDE CRITERIA EXIST?
ACUTE LETHALITY		
Oral	N}	Y}
Dermal	N}	Y}
Inhalation (Vapors)	}[6]	}[7]
aerosol	N}	Y}
dusts/ particles	N}	Y}
SKIN IRRITATION	N	Y[8]
SKIN SENSITIZATION (ANIMALS)	N	Y[9]
EYE IRRITATION	N	Y[10]
SUBCHRONIC (ORAL/DERMAL/INHALATION)	N	Y[11]
REPRODUCTION STUDY	N	Y[12]
DEVELOPMENTAL TOX	Y[13]	Y[14]

[6] 43 Fed Reg at 11114, comment 14:

"This policy statements directs the reporitng of specifiec effects when unknown to the Administrator. Many routine tests are based on a knowledge of toxicity associated with a chemicalL unknown effects occurring during such a range test may have to be reported if they are those of concern tot he Agency and if the information meets the criteria set forth in Parts V and VII."

[7] Guide at pp.22, 29-31.

[8] Guide at pp-34-36.

[9] Guide at pp-34-36.

[10] Guide at pp-34-36.

[11] Guide at pp-22; 36-37.

[12] Guide at pp-22

[13] 43 Fed Reg at 11112

"Birth Defects" listed.

[14] Guide at pp-22

NEUROTOXICITY	N	Y[15]
CARCINOGENICITY	Y[16]	Y[17]
MUTAGENICITY		
In Vitro	Y}[18]	Y} [19]
In Vivo	Y}	Y}
ENVIRONMENTAL		
Bioaccumulation	Y}	N
Bioconcentration	Y}[20]	N
Oct/water Part. Coeff.	Y}	N
Acute Fish	N	N
Acute Daphnia	N	N
Subchronic Fish	N	N
Subchronic Daphnia	N	N
Chronic Fish	N	N
AVIAN		
Acute	N	N
Reproductive	N	N
Reprodcutive	N	N

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[15]Guide at pp-23; 33-34.

[16]43 Fed Reg at 11112
"Cancer" listed

[17]Guide at pp-21.

[18]43 Fed Reg at 11112; 11115 at Comment 15
"Mutagenicity" listed/ *in vivo* vs *invitro* discussed; discussion of "Ames test".

[19]Guide at pp-23.

[20]43 Fed Reg at 11112; 11115 at Comment 16.

CAS# 9002-84-0
Chem: Polytetrafluoroethylene Resins
Title: Three-Week Feeding Study in Rats with Polytetrafluoroethylene Resins (Teflon®)
Date: 10/21/68
Summary of Effects: Enlarged liver when administered at 10% dietary concentration for 3 weeks.

THREE-WEEK FEEDING STUDY IN RATS WITH POLYTETRAFLUOROETHYLENE RESINS (TEFLON®)

Medical Research Project No. 1080

Haskell Laboratory Report No. 224-68

INTRODUCTION

It has previously been reported (Haskell Laboratory Report No. 49-60) that the feeding of an unsintered Teflon® 6 powder to rats at a dietary concentration of 25% produced slight liver enlargement after 90 days, but not after 14 and 21 days; the feeding of a diet containing 25% sintered Teflon® 6 did not cause liver enlargement under similar conditions. A subsequent report (Haskell Laboratory Report No. 56-61) indicated that the dietary administration to rats of 25% Teflon® 6c made with AHT, C_8-APFC or C_9-AFC as dispersing agents produced slightly enlarged livers after two and three weeks of continuous feeding; the addition of Teflon® made without dispersing agents to the diet of rats at a level of 25% did not cause liver enlargement. These data suggested that liver enlargement was attributable to the presence of dispersing agents in the Teflon®.

The present investigation was undertaken to determine whether the potential of polytetrafluoroethylene resins (PTFE) to produce enalrged livers in rats was dependent upon the dispersing agents used and/or the method of preparation. The two methods for preparing the various Teflon® samples were as follows: (1) the various PTFE resins were prepared at the Parkersburg, West Virginia, Plastics Plant and heated (i.e. removal of the volatile dispersing agents) before being shipped to Haskell Laboratory as fine powders; (2) unheated PTFE resins were received at the Plastics Department in the Experimental Station as five-gallon aqueous dispersions; the aqueous dispersions were frozen to effect partial coagulation and then evaporated to dryness at room temperature; the dried polymer was broken up and mixed with coagulated material obtained during freezing.

TEST MATERIALS

The test materials were received from the Plastics Department and were identified as shown in Table I.

PROCEDURE

Eighty ChR-CD male rats, 61 days old, were divided into eight equal average weight groups and fed the following diets and water ad libitum:

PROCEDURE (Cont'd.)

Group	Diet
I (Control)	Ground Purina Laboratory Chow (GPLC) + 1% Corn Oil (CO) + 10% Alphacel®*
II	GPLC + 1% CO + 10% TE-3238 (Plant)
III	GPLC + 1% CO + 10% T-6c (Plant)
IV	GPLC + 1% CO + 10% T-6 (Plant)
V	GPLC + 1% CO + 10% TD-37-X (Exp. Sta.)
VI	GPLC + 1% CO + 10% TE-3238 (Exp. Sta.)
VII	GPLC + 1% CO + 10% TE-5053 (Exp. Sta.)
VIII	GPLC + 1% CO + 10% T-42 (Exp. Sta.)

It had initially been planned to add the various PTFE resins to the diets at a concentration of 25%, in accordance with the procedure that had been employed in the earlier studies (Haskell Laboratory Reports Nos. 49-60 and 56-61). However, when the resins were added to the diet at the 25% level, in an initial study, it was observed that those rats that were to ingest the diets containing the resins that had been prepared through the aqueous dispersion phase did not eat as well as the control or the test rats receiving the plant products, and lost weight; when the concentration of the PTFE resins was lowered to 12.5%, the animals started to eat and gain weight. For this reason, the dietary level of the PTFE resins was initiated at the 10% level in the study herein reported. It is believed that the poor weight performances of the rats that received the Experimental Station products was due to the very high concentration of dispersing agents in these resins, which may have exerted a direct or toxic effect on the animal or may have affected the palatability of the diet so that it became unacceptable to the rats.

The animals were weighed twice a week; food consumption data were obtained once a week.

During the test, the animals were examined routinely for any abnormal behavior and any clinical manifestations of toxicity.

After 21 days of continuous feeding, five animals from each of the eight groups of animals were sacrificed; livers were removed, weighed, and then prepared for histopathologic evaluation. The remaining animals in each group were then placed on control diet (GPLC + 1% CO) and continued on this diet for 22 days; after this period of time, the animals were sacrificed and treated as above.

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* A product of the Nutritional Biochemicals Corporation, Cleveland, Ohio, composed of finely ground cellulose to supply non-nutritive bulk to diets.

RESULTS

1. Body Weight

A summary of the average weekly body weights is presented in Table II.

It would appear that the PTFE resins prepared at the Parkersburg Plant had no adverse effect upon the weight gain of the rats when incorporated into the diet at a 10% level. However, the PTFE resins prepared from the aqueous dispersions did depress the rate of weight gain slightly, as evidenced by the somewhat lower body weights of the rats during the test period and the slightly increased rate of weight gain during the recovery period when these resins were removed from the diet. A "t" test conducted at the end of the test and recovery periods indicated that only the rats in Group VIII, fed 10% T-42 (H-5669), had an average body weight that was significantly ($0.05 > p > 0.01$) lower than that of the controls at the end of the test period; there was no statistically significant difference between any test and control group at the end of the recovery period with respect to body weight.

2. Food Consumption

A summary of the average diet consumption data is presented in Table III.

Animals receiving 10% T-42 or 10% TD-37-X in the diet consumed less diet during the first week of the test period than did the control or other test groups. After this initial period, except for one group of rats, there was no meaningful difference among the various test groups and the control with respect to food consumption; during the third week of the test period, the animals receiving 10% T-42 consumed more diet than did any other group, apparently in an attempt to overcome the large deficit incurred during the first week.

In general, control and test groups consumed more diet during the test period than they did during the recovery period; this was attributed to the presence of 10% non-nutritive bulk in the diets during the test period.

3. Pathology of Liver

A summary of the individual and average liver weights and liver/body weight ratios is presented in Table IV.

Statistically significant increases in average liver weights were observed in Groups V-VIII (containing the PTFE resins obtained from the aqueous dispersions) at the end of the three-week test period. A series of "t" tests conducted at this time indicated a value of $p < 0.001$ for Groups V, VII, and VIII, and $0.05 > p > 0.01$ for Group VI. In addition, the slightly lower body weights for the animals in Group V, VI, and VII and the significantly lower body weights for the animals in Group VIII tended also to raise the liver/body weight ratios for these groups.

RESULTS (Cont'd.)

Only the animals in Group VII showed a statistically significant increase in liver weight at the end of the three-week recovery period ($0.01 > p > 0.001$); a decrease in body weight was not evident in this group.

Microscopic examination of the livers of animals that received the three PTFE resins that had been prepared at the Parkersburg Plant revealed no changes that could be attributed to their administration at either the end of the treatment period or the recovery period.

The liver cells of those animals that had received the PTFE resins that had been prepared from the aqueous dispersions showed microscopic changes in the cytoplasmic granules and vacuoles; at the end of the treatment period, these liver cells were pale and slightly enlarged and had a slightly granular cytoplasm. After a three-week recovery period, these liver cells had fewer vacuoles than the controls and, in the case of the animals in Group VII, fewer granules.

SUMMARY

The addition to rats' diets, at a 10% concentration, of polytetrafluoroethylene resins that had been prepared with chlorendic acid, C8-APFC, or C9-AFC as dispersing agents and subsequently heated, presumably at a high enough temperature to remove most of these volatiles, does not produce large livers; it would appear, therefore, that the small amount of residual dispersing agent (< 1 ppm) is without effect in this respect.

However, when these same polytetrafluoroethylene resins were prepared from dispersions in such a way that the dispersing agents remained, they did produce large livers when administered to rats at a 10% dietary concentration for three weeks; under these conditions, the concentration of dispersing agent was high enough, not only to produce these large livers, but also to exert a manifestly toxic effect, i.e., decrease rate of weight gain. The removal of these PTFE resins from the diet for three weeks was accompanied by a partial recovery from the hepatic effect, i.e., liver weights returned to normal, but a slight histologic change persisted.

It is also apparent from the data that PTFE resin obtained from a dispersion containing Duponol® contained enough residual agent (3.0%) to effect a manifestly toxic as well as hepatotoxic effect. These persisted throughout the three-week recovery period unlike the observations made with the PTFE resins that contained chlorendic, C8-APFC or C9-AFC. This difference may be related to the fact that Duponol® was present in the PTFE resin at a concentration that was approximately ten times greater than that for the other dispersing agents.

These results, showing that hepatomegaly in rats can be produced by low concentration of dispersing agents, suggest that dispersing agents, such as those described in this report, should be kept as low as possible consistent with good manufacturing practice. At present, lacking chronic toxicity data, we are not able to establish a "no-effect" level for these surfactants.

TABLE I

IDENTIFICATION OF PTFE RESINS

Sample Designation	Notebook Designation	Haskell No.	Dispersing Agent(s)	Wt. % Dispersing Agent and Additive in Resin at Time of Feeding	Method of Preparation
TE-3238	11210-113-G	5520	C_8-APFC	< 0.5*	Plant
			Chlorendic Acid	-	
T-6c	11210-113-F	5521	C_9-AFC	0.8*	Plant
T-6	11210-113-E	5522	C_8-APFC	0.5*	Plant
TD-37-X	11210-182-B	5666	C_8-APFC	0.27	Exp. Sta.
			Succinic Acid	0.18	
TE-3238	11210-182-C	5667	C_8-APFC	0.06	Exp. Sta.
			Chlorendic Acid	1.8	
TE-5053	11607-26	5668	Duponol®	3.0	Exp. Sta.
T-42	11210-182-A	5669	C_9-AFC	0.27	Exp. Sta.

* These figures given as parts per million (ppm).

TABLE II

SUMMARY OF AVERAGE WEEKLY BODY WEIGHTS IN GRAMS OF MALE RATS FED VARIOUS PTFE RESINS

Group or Sample Designation	Test Period (Days)				Recovery Period (Days)		
	0	7	14	21	7	14	22
I - Control	292	320	351	382	419	438	460
II - TE-3238 (Plant)	292	330	365	392	432	454	480
III - T-6c (Plant)	292	329	362	391	427	449	473
IV - T-6 (Plant)	293	326	362	388	430	452	473
V - TD-37-X (Exp. Sta.)	292	312	344	372	408	435	467
VI - TE-3238 (Exp. Sta.)	292	314	340	362	410	436	465
VII - TE-5053 (Exp. Sta.)	293	325	347	359	401	424	459
VIII - T-42 (Exp. Sta.)	293	249	304	349	394	427	452

TABLE III

SUMMARY OF AVERAGE DIET CONSUMPTION DATA IN GRAMS PER DAY OF MALE RATS FED VARIOUS PTFE RESINS

Group or Sample Designation	Test Period (Days)			Recovery Period (Days)		
	0-7	7-14	14-21	0-7	7-14	14-21
I - Control	26.8	28.0	28.3	25.3	24.3	23.1
II - TE-3238 (Plant)	28.7	29.9	30.2	26.8	27.0	26.6
III - T-6c (Plant)	28.9	30.5	30.2	27.2	26.0	25.0
IV - T-6 (Plant)	28.9	30.2	30.9	26.8	25.5	24.9
V - TD-37-X (Exp. Sta.)	23.4	30.1	30.3	26.7	26.0	25.5
VI - TE-3238 (Exp. Sta.)	26.7	29.1	30.0	28.4	27.8	27.1
VII - TE-5053 (Exp. Sta.)	28.0	31.4	32.9	32.0	27.6	27.8
VIII - 1-42 (Exp. Sta.)	13.7	27.7	35.0	28.0	28.2	26.4

TABLE IV

LIVER WEIGHTS AND LIVER/BODY WEIGHT RATIOS OF MALE RATS FED VARIOUS PTFE RESINS

Group or Sample Designation		Individual Liver Weights (g)		Individual Liver/Body Weight Ratios	
		After Test Period	After Recovery Period	After Test Period	After Recovery Period
I - Control		14.12	16.10	3.9	3.6
		13.95	21.03	4.0	4.4
		15.21	16.00	4.0	3.8
		14.00	17.14	3.8	3.5
		15.70	18.91	4.1	4.0
	Avg.	14.60	17.84	4.0	3.9
II - TE-3238 (Plant)		14.85	22.01	4.3	4.5
		15.35	16.00	4.0	3.5
		14.81	18.42	4.0	4.0
		14.10	26.20	3.8	4.8
		17.85	18.42	4.2	4.1
	Avg.	15.39	20.21	4.1	4.2
III - T-6c (Plant)		13.37	17.46	3.8	3.8
		17.70	16.30	4.3	3.6
		13.80	16.86	3.8	3.4
		17.50	19.98	4.4	4.4
		15.13	18.72	3.9	3.7
	Avg.	15.50	17.86	4.0	3.8
IV - T-6 (Plant)		12.94	16.36	3.8	3.6
		16.70	16.73	4.3	3.7
		15.61	18.05	4.2	3.8
		15.55	18.07	3.9	4.0
		15.10	18.76	3.8	3.6
	Avg.	15.18	17.59	4.0	3.7
V - TD-37-X (Exp. Sta.)		29.99	21.60	7.6	4.6
		21.07	19.91	6.6	4.3
		24.00	19.22	6.4	4.2
		24.45	19.25	6.6	4.2
		23.80	21.63	6.3	4.5
	Avg.	24.66	20.32	6.7	4.4

TABLE IV (Cont'd.)

Group or Sample Designation	Individual Liver Weights (g)		Individual Liver/Body Weight Ratios	
	After Test Period	After Recovery Period	After Test Period	After Recovery Period
VI - TE-3238 (Exp. Sta.)	16.60	19.45	5.2	4.1
	14.27	21.38	4.2	4.7
	16.21	17.49	4.4	3.8
	18.20	21.50	5.1	4.7
	18.00	19.30	4.9	4.0
Avg.	16.65	19.82	4.8	4.3
VII - TE-5053 (Exp. Sta.)	22.50	24.28	6.1	5.5
	22.61	22.38	6.6	5.1
	22.25	29.68	6.6	5.9
	19.22	23.00	5.9	5.0
	25.00	25.70	6.9	5.7
Avg.	22.32	25.01	6.4	5.4
VIII - T-42 (Exp. Sta.)	27.28	22.98	7.5	5.0
	26.10	16.37	7.5	3.6
	23.50	20.90	7.8	4.5
	25.65	17.72	7.7	4.0
	24.00	19.08	7.2	4.3
Avg.	25.31	19.41	7.5	4.3

Triage of 8(e) Submissions

Date sent to triage: _____ NON-CAP (CAP)

Submission number: 12219A TSCA Inventory: (Y) N D

Study type (circle appropriate):

Group 1 - Dick Clements (1 copy total)

ECO AQUATO

Group 2 - Ernie Falke (1 copy total)

ATOX (SBTOX) SEN w/NEUR

Group 3 - Elizabeth Margosches (1 copy each)

STOX CTOX EPI RTOX GTOX

STOX/ONCO CTOX/ONCO IMMUNO CYTO NEUR

Other (FATE, EXPO, MET, etc.): _____

Notes:

THIS IS THE ORIGINAL 8(e) SUBMISSION; PLEASE REFILE AFTER TRIAGE DATABASE ENTRY

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entire document: (0) 1 2 **pages** 1,9 **pages** _____

Notes:

Contractor reviewer : JW **Date:** 1/17/96

CECATS\TRIAGE TRACKING DBASE ENTRY FORM

CECATS DATA: Submission # SEHQ-1092-12219 SEQ. A

TYPE: (INT.) SUPP FLWP

SUBMITTER NAME: E. I. Dupont de Nemours and Company

SUB. DATE: 10/15/92 OTS DATE: 10/27/92 CSRAD DATE: 04/04/95

INFORMATION REQUESTED: FLWP DATE:
0501 NO INFO REQUESTED
0502 INFO REQUESTED (TECH)
0503 INFO REQUESTED (VOL ACTIONS)
0504 INFO REQUESTED (REPORTING RATIONALE)

DISPOSITION:
(0539) REFER TO CHEMICAL SCREENING
(0570) CAP NOTICE

VOLUNTARY ACTIONS
(0401) NO ACTION REPORTED
0402 STUDIES PLANNED/UNDERWAY
0403 NOTIFICATION OF WORKER EXPOSURE
0404 LABEL/MSDS CHANGES
0405 PROCESS/HANDLING CHANGES
0406 APP/USE DISCONTINUED
0407 PRODUCTION DISCONTINUED
0408 CONFIDENTIAL

CHEMICAL NAME: Teflon 6

CAS#: 9002-84-0

INFORMATION TYPE:		P F C
0201	ONCO (HUMAN)	01 02 04
0202	ONCO (ANIMAL)	01 02 04
0203	CELL TRANS (IN VITRO)	01 02 04
0204	MUTA (IN VITRO)	01 02 04
0205	MUTA (IN VIVO)	01 02 04
0206	REPRO/TERATO (HUMAN)	01 02 04
0207	REPRO/TERATO (ANIMAL)	01 02 04
0208	NEURO (HUMAN)	01 02 04
0209	NEURO (ANIMAL)	01 02 04
0210	ACUTE TOX. (HUMAN)	01 02 04
0211	CHR. TOX. (HUMAN)	01 02 04
0212	ACUTE TOX. (ANIMAL)	01 02 04
(0213)	SUB ACUTE TOX (ANIMAL)	01 (02) 04
0214	SUB CHRONIC TOX (ANIMAL)	01 02 04
0215	CHRONIC TOX (ANIMAL)	01 02 04

INFORMATION TYPE:		P F C
0216	EPI/CLIN	01 02 04
0217	HUMAN EXPOS (PROD CONTAM)	01 02 04
0218	HUMAN EXPOS (ACCIDENTAL)	01 02 04
0219	HUMAN EXPOS (MONITORING)	01 02 04
0220	ECO/AQUA TOX	01 02 04
0221	ENV. OCCC/REL/FATE	01 02 04
0222	EMER INCI OF ENV CONTAM	01 02 04
0223	RESPONSE REQUST DELAY	01 02 04
(0224)	PROD/COMP/CHEM ID	01 02 04
0225	REPORTING RATIONALE	01 02 04
0226	CONFIDENTIAL	01 02 04
0227	ALLERG (HUMAN)	01 02 04
0228	ALLERG (ANIMAL)	01 02 04
0239	METAB/PHARMACO (ANIMAL)	01 02 04
0240	METAB/PHARMACO (HUMAN)	01 02 04

INFORMATION TYPE:		P F C
0241	IMMUNO (ANIMAL)	01 02 04
0242	IMMUNO (HUMAN)	01 02 04
(0243)	CHEM/PHYS PROP	01 02 04
0244	CLASTO (IN VITRO)	01 02 04
0245	CLASTO (ANIMAL)	01 02 04
0246	CLASTO (HUMAN)	01 02 04
0247	DNA DAM/REPAIR	01 02 04
0248	PROD/USE/PROC	01 02 04
0251	MSDS	01 02 04
0299	OTHER	01 02 04

TRIAGE ENTRY

NON-CBI INVENTORY
(YES)
NO
IN PLANNING

ONGOING REVIEW
YES (DROP/REFER)
NO (CONTINUE)
REFER

SPECIES
RAT

TOXICOLOGICAL CONCERN:
(LOW)
MED
HIGH

USE:

PRODUCTION:

CAS SR

COMMENTS

12219A

L

TE-3238: Subacute oral toxicity in rats is of low concern. Ten male ChR-CD rats received 6,000 mg/kg/day (converted from 10% dietary concentration) for 21 days. There were no effects on liver weight or liver histopathology.

L

T-6c: Subacute oral toxicity in rats is of low concern. Ten male ChR-CD rats received 6,000 mg/kg/day (converted from 10% dietary concentration) for 21 days. There were no effects on liver weight or liver histopathology.

L

T-6: Subacute oral toxicity in rats is of low concern. Ten male ChR-CD rats received 6,000 mg/kg/day (converted from 10% dietary concentration) for 21 days. There were no effects on liver weight or liver histopathology.

L

TD-37-X: Subacute oral toxicity in rats is of low concern. Ten male ChR-CD rats received 6,000 mg/kg/day (converted from 10% dietary concentration) for 21 days. Average liver weight was significantly increased at three weeks, but returned to normal following the recovery period. Histopathological examination revealed alterations to the cytoplasmic granules and vacuolation. After treatment, these liver cells were pale and slightly enlarged and had a slightly granular cytoplasm. After the recovery period, these cells had fewer vacuoles.

L

TE-3238: Subacute oral toxicity in rats is of low concern. Ten male ChR-CD rats received 6,000 mg/kg/day (converted from 10% dietary concentration) for 21 days. Average liver weight was significantly increased at three weeks, but returned to normal following the recovery period. Histopathological examination revealed alterations to the cytoplasmic granules and vacuolation. After treatment, these liver cells were pale and slightly enlarged and had a slightly granular cytoplasm. After the recovery period, these cells had fewer vacuoles.

L

TE-5053: Subacute oral toxicity in rats is of low concern. Ten male ChR-CD rats received 6,000 mg/kg/day (converted from 10% dietary concentration) for 21 days. Average liver weight was significantly increased after three weeks of treatment and after the recovery period. Histopathological examination revealed alterations to the cytoplasmic granules and vacuolation. After treatment, these liver cells were pale and slightly enlarged and had a slightly granular cytoplasm. After the recovery period, these cells had fewer vacuoles and granules.

L

T-42: Subacute oral toxicity in rats is of low concern. Ten male ChR-CD rats received 6,000

mg/kg/day (converted from 10% dietary concentration) for 21 days. At the end of the test period, the rats exhibited significantly decreased body weight. Average liver weight was significantly increased at three weeks, but returned to normal following the recovery period. Histopathological examination revealed alterations to the cytoplasmic granules and vacuolation. After treatment, these liver cells were pale and slightly enlarged and had a slightly granular cytoplasm. After the recovery period, these cells had fewer vacuoles.